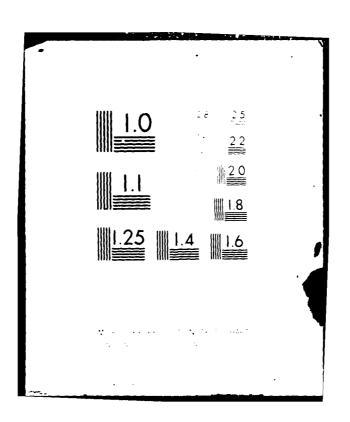
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DOMESTIC SWINE IN PHYSIOLOGICAL RESEARCH

III. Blood Gas and Acid-base Values of Arterial and Venous Blood from Young Anesthetized Pigs Maintained under Steady-state Ventilatory Conditions

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DIVISION OF COMBAT CASUALTY CARE





LETTERMAN ARMY INSTITUTE OF RESEARCH PRESIDIO OF SAN FRANCISCO

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Domestic Swine in Physiological Research. 111. Blood Cas and Acid-base Values of Arterial and Venous Blood from Young Anesthetized Pigs Maintained under Steady-state Ventilatory Conditions --Hannon and Moores

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ABSTRACT

The arterial and venous blood gas and acid-base characteristics of anesthetized, young domestic swine were determined under steady-state ventilatory conditions designed to establish arterial P O2 at 100 torr and pH at 7.40. Under these circumstances, mean (N=15) femoral artery values were: P O2, 97 torr; S O2, 94%; C O2, 15.4 ml/dl; pH, 7.399; P CO_2 , 47 torr; [HCO3], 27.6 mEq/l; and [B.E.], 2.8 mEq/l. Values for pulmonary artery, mixed venous blood were: P O2, 36 torr; SaO2, 51%; $C O_2$, 8.5 ml/dl; pH, 7.335; P CO_2 , 57 torr; [HCO3], 29.6 mEq/1; and [B.E.], 3.9 mEq/1. Comparisons of venous values obtained from various vascular sites (pulmonary artery, anterior vena cava, posterior vena cava, internal jugular vein, femoral vein and coronary sinus) revealed numerous between-vessel differences in blood gas and acid-base status. The arterial characteristics of this anesthetized preparation differed from those of the conscious pig; the anesthetized animal had lower pH, [HCO3] and [B.E.] values and higher P O2 and P CO2 values than the conscious animal. Anesthesia with mechanical ventilation appeared to produce defects in alveolar-arterial gas exchange similar to those reported for other species. The anesthetized pig, nevertheless, can serve as an effective biomedical model for human oriented research.

PREFACE

This is the third in a series of studies concerned with the utility of domestic swine as a large animal biomedical model for human-oriented physiological experiments. Earlier reports provided a brief review of the use of pigs in medical research and a description of the cation and anion characteristics of porcine arterial blood. Future reports will be concerned with the surgical preparation of swine for experiments to be conducted under conscious conditions and with the population characteristics for the blood gas and acid-base status of arterial blood obtained under basal conditions.

We would like to express our appreciation to Virginia L. Gildengorin, Ph.D., for the invaluable assistance she provided in the statistical evaluation of the data reported here, to Lottie B. Applewhite for the many editorial and format improvements incorporated in this report, and especially to Linda Pukajlo for the innumerable hours she spent typing, proofreading, and assembling the manuscript.

TABLE OF CONTENTS

Abs	tract	•	•		•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•		•	•	•	i
Pre	face		•		•				•	•	•			•	•	•												•	ii
Tab	le of	Cc	nt	er	its	š	•	•		•		•	•	•				•			•		•			•			iii
BOD	Y OF I	ÆF	POF	T																									
	INTRO	JDU	CI	'IC	N								•	•			•		•			•							1
	METHO	DDS	3	•	•		•	•	•			•		•				•	•		•								2
	RESUI	LTS	5	•	•				•	•								•		•			•		•			•	3
	DISC	JSS	SIC	N		•	•	•		•	•	•	•				•				•		•		•		•	•	4
	CONC	ւՄՏ	SIC	NS	5	•	•		•	•		•	•	•					•				•	•					9
	RECO	ME	ENE	PAG	IC)NS	3	•		•				•		•		•		•	•			•	•	•	•		10
	REFE	REN	ICE	s	•		•	•	•			•			•	•	•	•	•	•	•		•	•	•				11
App	endix	(1	(ak)le	28	1-	-1:	L)	•	•		•		•	•		•	•	•			•		•	•	•			15
Dis	tribut	tic	'n	Li	st	•	_		_				_		_		_			_					_			_	28

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DOMESTIC SWINE IN PHYSIOLOGICAL RESEARCH. III. Blood Gas and Acid-base Values of Arterial and Venous Blood from Young Anesthetized Pigs Maintained under Steady-state Ventilatory Conditions

Historically, the mongrel dog has served as the predominant large animal model for experimental studies of mammalian cardiovascular function. Such predominance continues at the present time, but with increasing frequency one sees miniature or domestic pigs being used in the conduct of these studies. In most instances, the selection of swine as a biomedical model stems from both anatomical and functional considerations. The anatomy of the coronary vasculature in the pig, for example, more closely resembles that of man than does the coronary vasculature of the dog (1,2). As a consequence, the pig is considered by some to be a superior animal model for studies of coronary blood flow (3,4) and myocardial function (4-7). The domestic pig would also appear to be superior to the dog for investigations of circulatory shock. Hepatic venous outflow in the dog, for example, is regulated by a highly developed sphincter-like mechanism (8) which is quite sensitive to the vasoactive materials produced during shock (9). Closure of this sphincter mechanism in the shocked dog results in hepatic congestion, increased portal vein pressure, and intestinal hemorrhage (9). These responses are rarely seen in humans or swine (9, 10) probably because the sphincter mechanism is poorly developed (11).

As indicated in earlier reports of this series (12, 13), effective use of the domestic pig as a biomedical model is oftentimes encumbered by a lack of readily available experimental data describing the population characteristics for certain physiologic and biochemical variables. To address one such problem, we measured the major arterial anion and cation concentrations in young anesthetized domestic swine maintained under steady state ventilatory conditions, and these values were compared to those published in the literature (13). A paucity of data was encountered also when we attempted to ascertain normal values for the blood gas and acid-base status of these animals. Most values reported in the literature were measured in venous blood samples with little or no attention given

to the physiologic status of the pigs at the time of blood collection. In view of the many factors (eg. ventilation, kidney function, sampling site) that can affect blood gas and acid-base status, it was perhaps not too surprising to find wide divergence in the values obtained by various investigators.

The study reported here had two objectives. The first was to determine whether or not the anesthetized domestic pig could be maintained under steady state ventilatory conditions such that arterial pH and PO_2 were established at levels commonly seen in humans, that is, about 7.40 and 100 torr respectively. The second objective was to provide, in the same animal model, data on the blood gas and arterial and venous samples obtained from vascular sites that are commonly monitored in cardiovascular research studies.

METHODS

Fifteen young domestic pigs (Hampshire - Duroc Cross) of both sexes, 60 to 90 days old, were used. They were obtained from a commercial breeder (J.G. Boswell, Corcoran CA) and, upon receipt at the laboratory, were maintained in a common indoor holding area until utilized in the study, usually within two weeks after arrival. They were fed a commercial ration (Purina Pig Chow, Ralston-Purina Co. St. Louis, MO) and received water ad libitum.

After an overnight fast, each pig was brought into the laboratory and anesthetized. Halothane was used for induction, meperidine hydrochloride and nitrous oxide for maintenance, and succinylcholine for paralysis. The animal was placed in a supine position, and ventilation was provided by means of an endotracheal tube and a mechanical ventilator (Ohio Medical Products, Madison, WI). The latter was used also to regulate nitrous oxide and oxygen delivery. Ventilation and oxygen delivery were adjusted to establish an arterial P O; of approximately 100 torr and a pH of approximately 7.40. This was ascertained by periodic sampling of femoral artery blood obtained by means of an indwelling catheter. No infusions of acid or base were required to maintain steady-state acidity levels. Normothermia (37C) was maintained by means of a circulating warm water heating pad. The pig remained under these conditions for approximately one hour, during which a sternotomy was performed and indwelling catheters were placed in the femoral vein, anterior and posterior vena cava, pulmonary artery, internal jugular vein, and coronary sinus.

When P O_2 and pH had been stabilized for at least one-half hour without any adjustments of pulmonary minute volume or inspired O_2 tension, 3 ml samples of blood were drawn from each of the catheters for blood gas and acid-base determinations. The total time involved

in obtaining these samples was 30 seconds or less. Heparin (1000 units/ml) was used to fill the dead space within the syringes, and care was exercised in avoiding sample contamination with air bubbles. Immediately after withdrawal the syringes were capped and immersed in ice water where they remained until the blood gas and acid-base measurements were completed. These measurements were made with an automated blood gas analyzer (Model 813, Instrumentation Laboratory, Lexington, MA), which had been calibrated with precision buffers and analyzed gases provided by the manufacturer. In addition, hemoglobin (Hb) concentration was determined with a cooximeter (Instrumentation Laboratory, Lexington, MA), and the oxygen saturation (S O2) of all samples was measured with an oximeter (American Optical Co., Buffalo, NY). Oxygen content (ml/dl of blood) was calculated as the product of 1.34 (ml O_2 /gHb) X Hb (g/dl) X SO_2 (%) X 10^{-2} . All data were summarized in terms of the mean population value, standard deviation, standard error of the mean, and range. The significance of differences in venous values were evaluated with single-factor analyses of variance. Interrelationships between variables were evaluated with Pearson Product-Moment correlation values. RESULTS

RESULTS

Body weight, hemoglobin, and ventilatory functions recorded at the time of blood sampling are summarized in Table 1. Tables 2 through 8 summarize the blood gas and acid-base data of samples collected from various arterial and venous sites. These data show that reasonable success was achieved in establishing a ventilatory steady state in which the arterial (femoral) pH was maintained at about 7.40 and P O_2 at about 100 torr.

In viewing these tables, marked differences in the blood gas and acid-base characteristics of arterial and venous blood, including mixed-venous blood from the pulmonary artery, are readily evident. Not so readily evident are the differences between venous blood samples. Analyses of variance evaluation thus revealed significant between-vessel differences for all of the venous blood gas and acid-base variables except bicarbonate (Tables 9a, 9b). In general, more between-vessel differences were found for oxygen variables than for CO₂ or other acid-base variables.

Table 10 summarizes the possible interrelationships between key blood gas and acid-base variables for arterial and mixed venous blood. Significant correlation coefficients for certain of these interrelationships, such as pH, P $\rm CO_2$, [HCO $_3$] and [B.E.] might be anticipated, since they all reflect characteristics of a common functional entity. Others, however, were unexpected. The latter included the significant positive correlation coefficients that were obtained between P $\rm O_2$ and the P $\rm CO_2$, bicarbonate

concentration and base excess concentration of blood taken from the femoral artery: these correlations were not significant in mixed venous blood taken from the pulmonary artery.

Possible correlations between ventilatory variables and key arterial blood gas and acid-base variables are summarized in Table 11. A highly significant positive correlation was found between arterial P O2 and the percentage oxygen in the inspired air (F_1O_2) , an effect that would be expected. But unexpectedly, a highly positive correlation was found between P CO2 and F_1O_2 and, albeit of borderline significance, between F_1O_2 and bicarbonate concentration. Respiratory frequency also showed a positive correlation of borderline significance to arterial P CO2. Inspiratory minute volume and tidal volume showed no significant relationships to blood gas and acid-base status, although the negative correlation between P CO2 and tidal volume approached statistical significance.

DISCUSSION

The results of this study show that it is indeed possible to establish steady state ventilatory conditions in which the values for arterial pH and P O of anesthetized swine simulate those of the normal human. The recorded data reveal, however, two potential problems that must be addressed if the present simulation model is used for human-oriented investigations. One of these was the high porcine values, relative to those characteristic of humans (14, 15) for arterial and venous P CO₂, bicarbonate, and base excess. The other problem concerned potential distortion of normal alveolar-pulmonary capillary gas exchange by the mechanical ventilation procedures used to establish steady state values for pH and P O₂.

Were the elevated acid-base values recorded in this study truly characteristic of the normal pig or were they the result of the experimental conditions imposed on the animal before the blood gas and acid-base measurements were made? This question can not be addressed by the experimental work reported here, and data reported in the literature, for the most part, fail to resolve the issue. Reported porcine venous values for pH are higher (16-24), lower (18, 25-27), or about the same (18, 20, 25, 26, 28-30) as those obtained in the present study. Most reported values for venous P CO_2 are lower than those recorded in the present study (16-20, 22, 24, 26-30), but frequently equivalent values are seen (18, 20, 23, 25). Venous bicarbonate and base excess concentrations reported in the literature, relative to those reported here, show about the same variability as P CO_2 .

On the arterial side, far fewer blood gas and acid-base measurements have been reported. In general, it would appear that most of

the reported pH values (18, 21, 23, 28) are higher than those recorded here, but occasional exceptions are seen (31). In contrast, most reported P $\rm CO_2$ values (18, 21, 28, 31), again with occasional exceptions (18), are lower than those recorded here. Measurements of bicarbonate and base excess concentrations have been so few and the reported data so divergent (18, 23, 28) that it is nearly impossible to discern characteristics of the normal pig from published values.

Inconsistencies in the published values for the blood gas and acid-base characteristics of the normal pig can be attributed to a variety of factors. Foremost among these were disparities in the physiological status of the animal at the time blood samples were taken for analysis. In almost every instance, control or supposedly normal values were obtained as a prelude to some experimental manipulation (e.g. exercise, heat stress, shock) of immediate interest to the investigator. In most instances, there is little evidence that steady-state metabolic and ventilatory conditions were established before these control measurements were made. Local or general anesthetics, or physical restraint were oftentimes employed to secure the requisite blood samples for study. These manipulations, obviously, can readily alter the physiological status of the animal and consequently the outcome of blood gas and acid-base measurements. Other variables include differences in animal size, environmental temperature at the time of study, animal breed, and, in the case of venous blood, the vascular sampling site. The distinctly different blood gas and acid-base characteristics recorded here for two commonly used veins, the jugular and the femoral, illustrate this last point.

Ideally, control or normal values should approximate those obtained under one widely-recognized standard condition. The basal metabolic state (i.e. the metabolic state characteristic of a recumbent, well-rested, conscious subject who has been fasted overnight) represents, perhaps, the most common reference standard. Few attempts have been made to acquire data from pigs in this condition. This is understandable since most pigs are excitable animals, and some sort of physical or chemical restraint is usually required before experimental measurements can be made. One way to minimize, or eliminate, the restraint variable is to obtain blood samples from the conscious animal by means of chronically implanted vascular catheters. A few investigators have reported blood gas and acid-base values for samples so acquired (18, 21, 23, 28), but, unfortunately, their reports contain little information about the metabolic state of the animals at the time of study.

Basal conditions were approached, if not achieved, in a recent investigation of hemorrhagic hypotension in conscious page (32). In this study, the animals received chronically-implanted arterial catheters 7 to 10 days before they were subjected to experimentation.

At the time control samples were taken for blood gas and acid-base evaluation, they had been faster overnight and had been in a conscious, unrestrained, recumbent position for 30 to 60 minutes before any measurements were made. Under these conditions, 18 pigs had an average pH of 7.50, a P $\rm CO_2$ of 40.3 torm, a P $\rm O_2$ of 81.6 torm, bicarbonate concentration of 30.4 mEq/1, and a base excess concentration of 7.5 mEq/1. An untreated control group (N=6) included in this study showed little deviation from the foregoing values when the measurements were repeated at hourly intervals over a 6-hour period.

All of the blood gas and acid-base values obtained in the present investigation differ appreciably from values previously recorded in conscious pigs. In large measure these differences appear attributable to the establishment of a steady-state arterial ph at 7.40. This resulted in P CO, values that were higher and [HCOT] values that were lower than those observed in the conscious animal. The bicarbonate difference, furthermore, appeared responsible, at least in part, for the lower base excess values reported here, between the two studies the arterial bicarbonate difference was 2.8 mEq/1, the base excess difference 5.7 mEq/1. Additionally, arterial P O; in the conscious animal is considerably lower than the 100 torr steady-state value selected for the present study. The value in the conscious pig, furthermore, was lower than the value usually seen in the conscious human (14, 33). Arterial oxygen saturation, it might be noted, is lower also because of the rightward position of the porcine oxyhemoglobin dissociation curve relative to that of man (34).

In view of the forgoing, it is clearly evident that researchers using anesthetized swine as a biomedical model must weigh the merits of establishing steady-state blood gas and acid-base conditions which simulate those of humans against those which simulate the normal conscious pig.

The second potential problem raised by this study may have more serious consequences if the biomedical model described here is to be used for routine cardiovascular or other studies. The problem centers on the oxygen fraction of inspired air that was needed to maintain arterial P O_2 at the desired level (Table 1), and the positive correlation of arterial P O_2 and P CO_2 that was obtained once steady state conditions were established. Taken together, these observations suggest that the selected ventilatory and arterial parameters utilized in this study may lead to an impairment of normal alveolar-arterial gas exchange.

Support for this implication is found when the alveolar to arterial (A-a) oxygen gradient of these pigs is compared to those of conscious humans or conscious pigs. Since alveolar oxygen tension (P_AO_7) values were not obtained in the present study nor,

insofar as can be determined, in other studies of swine, these comparisons must be based on estimated values. Accordingly, if certain assumptions are made, $P_{A}O_{2}$ can be calculated from the alveolar gas equation (see Otis (35) for derivation):

$$P_{A}O_{2} = P_{I}O_{2} - \frac{P_{A}CO_{2}}{R} + F_{I}O_{2} \frac{P_{A}CO_{2}}{R} - P_{A}CO_{2}$$

For present circumstances, it will be assumed that $F_1O_2=0.209$ and $P_1O_2=149$ torr for conscious humans and pigs breathing air at sea level. The average steady-state F_1O_2 for the anesthetized pigs in this study was 0.380 which would be equivalent to a P_1O_2 value of 271 torr. Since CO_2 has a high membrane diffusion capacity (36) equality of P_1CO_2 and P_2CO_2 will be assumed (i.e. 40 torr for conscious pigs and humans and 47 torr for anesthetized pigs). Finally, an R value of 0.80 will be assumed for all conditions, and measured (32) or commonly reported (14) values for P_2O_2 will be used in the calculations of P_2O_2 . The alveolar equation thus implemented yields the following characteristics for swine and humans:

	P _A O ₂	P _O	PAO2-PAO2
Conscious human	101	90	11
Conscious pig	101	82	19
Anesthetized pig	217	97	120

On the basis of this tabulation it is readily evident that the A-a gradient of the anesthetized pigs used in this study was approximately 6 times that of the conscious pig and 10 times that of the conscious human. The accuracy of these A-a gradient estimations, obviously, hinges on the accuracy of the various assumed values which were inserted into the alveolar gas equation. Of these, the most likely deviation would be the assumed P_ACO_2 values; they may have been considerably lower then P_aCO_2 values. A lower P_ACO_2 value, however, would lead a higher calculated value for P_AO_2 and, consequently, a higher A-a gradient. Thus, if P_ACO_2 were 40 torr in anesthetized pigs, the calculated P_AO_2 would be 225 torr and the A-a gradient 128 torr.

The factors responsible for the positive correlation between steady-state values for P_aO_2 and P_aCO_2 seen here (Table 10) were not investigated. Under normal circumstances one would expect a negative correlation between the two variables. For example, if ventilation were increased by voluntary or mechanical means then P_aO_2 should increase also while P_aCO_2 should decrease. The existence of a positive correlation, therefore, suggests the presence of an impediment to normal alveolar-arterial exchange of both oxygen and carbon dioxide. In this regard, it is important to note that a positive correlation was not obtained for venous blood taken from the pulmonary artery, or for that matter for blood from any other venous site. The effect, therefore, arose during the transit of blood through the lungs.

In the establishment of steady-state values for pH_a and P_aO_2 , both $F_{I}O_{2}$ and ventilatory minute volume were manipulated. If preliminary measurements showed PaO2 values to be too low then the F_1O_7 of the inspired air was increased. The opposite maneuver was used if PaO, values were too high. Similarly, if preliminary pHa values were too low (or high) ventilation would be increased (or decreased) to enhance (or attenuate) carbon dioxide elimination and so shift arterial pH values in the desired direction. After steady state conditions had been established, the selected fraction of oxygen in the inspired air had a much greater impact on arterial blood gas and acid-base status than the selected level of ventilation. Thus, P_aO_2 showed, as would be anticipated, a positive correlation to F₁O₂ (Table 11). But unexpectedly, F₁O₂ also showed a highly positive correlation with PaCO2. Under steady-state conditions one would expect these two variables to be unrelated. One might anticipate also a negative correlation between ventilatory minute volume or tidal volume and PaCO2. The latter was not seen, however (Table 11).

What could account for these apparently paradoxical results? An impediment to normal alveolar-pulmonary capillary gas exchange, incurred during the establishment of the steady state, would seem to be one possibility. If such an impediment occurred shortly after the animal was placed on the mechanical ventilator, then PaO2 would tend to decrease while PaCO2 would tend to increase. The increase in PaCO, would be a desired effect since it would be required in the establishment of steady-state pHa at 7.40. That is, if the pH_a was about 7.50 when the pig was conscious (32), then a P_aCO_2 increase would be needed to lower the value to 7.40 when it was anesthetized. In most instances, defects in alveolar-arterial gas exchange should have a much greater impact on PaO2 than on P_aCO_2 , because CO_2 , compared to O_2 , has a 20-fold greater alveolar membrane solubility and proportionately greater membrane diffusion capacity (36). In the present study, therefore, sizable increases in F_IO₂ level were required to achieve the desired steady-state

level for P_aO_2 . These increments, it might be assumed, should have had no effect on P_aCO_2 since the latter was regulated by ventilatory changes. This assumption, however, may not be valid since Larson and Severinghaus (37) showed in man that increases in F_1O_2 led to increases in the a-A gradient for CO_2 . They attributed the gradient increase to an oxygen-induced relaxation of the pulmonary vascular bed which diverted blood flow to the more poorly ventilated portions of the lung. Such a mechanism, if operative in the anesthetized pig, could account for the positive correlation of P_aO_2 and P_aCO_2 seen in the present study. Accordingly, greater increments in F_1O_2 would produce proportionately greater increments in P_aO_2 (Table 11), but they would also produce proportionately greater increments in the a-A gradient for CO_2 and proportionately greater increments in P_aCO_2 (Table 11).

Defects in pulmonary gas exchange during anesthesia have been widely recognized. In general, they have been attributed to an uneven distribution of alveolar ventilation in relation to alveolar volume and blood flow (38). Anesthetics, including nitrous oxide, cause atelectasis (39), a decrease in pulmonary compliance (40), an increase in dead space (41), venous admixture to arterial blood in the lung with a lowering of P_{aO_2} (39) and an increase in the a-A gradient for CO_2 (41). It appears that most of these effects can be attributed to an unchanging tidal volume and decreased residual functional capacity, since equivalent defects have been recorded in conscious, mechanically ventilated humans (42).

The problems with pulmonary gas exchange encountered in the present study, therefore, would not seem to be unique to swine and should not seriously detract from their use in biomedical studies requiring anesthetized animals. In fact, this same preparation has been used successfully for extracorporeal circulation experiments lasting several hours (4).

CONCLUSIONS

- With proper regulation of ventilation and inspired oxygen tension, the arterial P O₂ and pH of anesthetized swine can be brought to steady-state values that approximate those of humans.
- Ounder these circumstances the values for arterial P CO₂, [HCO₃] and P O₂ are higher than those seen in conscious swine. Base excess concentration and pH values are lower than those seen in conscious swine.

- Ounder steady-state ventilatory conditions there are significant, between-vessel, differences in the blood gas and acid-base characteristics of venous blood.
- Mechanical ventilation in the anesthetized pig leads to impairment of normal alveolar-arterial gas exchange.

RECOMMENDATIONS

- o The feasibility of establishing blood gas and acid-base characteristics of the anesthetized pig at values approximating those seen in the conscious pig should be investigated.
- o In anesthetized mechanically ventilated swine, attempts should be made to minimize or eliminate defects in alveolar-arterial gas exchange by the inclusion of periodic hyperinflation.

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LEGENDS OF TABLES

- TABLE 1 Animal Characteristics and Experimental Conditions at the Time of Blood Gas and Acid-Base Measurement
- TABLE 2 Arterial and Venous P O2 Values for Young Swine
- TABLE 3 Arterial and Venous O2 Saturation Values for Young Swine
- TABLE 4 Arterial and Venous O2 Content Values for Young Swine
- TABLE 5 Arterial and Venous pH Values for Young Swine
- TABLE 6 Arterial and Venous P CO2 Values of Young Swine
- TABLE 7 Arterial and Venous Bicarbonate Values for Young Swine
- TABLE 8 Arterial and Venous Base Excess Values for Young Swine
- TABLE 9a Analyses of Variance Evaluation of Differences in Blood Gas and Acid-Base Characteristics of Venous Blood Taken from Various Vascular Sites of Young Pigs
- TABLE 9b Newman-Keuls Evaluation of Differences in Blood Gas and Acid-Base Characteristics of Venous Blood Taken from Various Vascular Sites of Young Pigs
- TABLE 10 Correlation Matrix for Arterial and Mixed Venous Blood Gas and Acid-Base Variables of Young Swine
- TABLE 11 Correlation of Femoral Artery Blood Gas and Acid-Base Variables to Ventilatory Variables of Young Swine

APPENDIX

TABLE 1

Animal Characteristics and Experimental Conditions at the Time of Blood Gas and Acid-Base Measurement

Characteristic	Mean	Range	s.D.	SEM
Body Weight (kg)	29.7	22.7-40.0	5.92	1.53
Hemoglobin (g/dl)	12.3	11.0-13.1	0.73	0.19
Inspired O_2 (%)	38	30-50	7.6	3.1
Insp. Min. Vol (1/min, BTPS)	6.5	3.5-8.4	1.38	0.36
Resp Freq. (breaths/min)	15	11-18	1.8	0.5
Tidal Vol. (ml/BTPS)	426	320- 550	72	19

TABLE 2 $\label{eq:arterial} \mbox{Arterial and Venous P O}_{2} \mbox{ Values* for Young Swine}$

Vessel	Mean	Range	S.D.	S.E.M.
Femoral Artery	97	86-115	8.6	2.2
Pulmonary Artery	36	26- 44	4.2	1.1
Ant. Vena Cava	39	33- 47	4.2	1.1
Post. Vena Cava	35	26- 42	4.4	1.1
Int. Jugular Vein	39	29- 51	6.4	1.7
Femoral Vein	35	25- 44	4.7	1.2
Coronary Sinus	30	22- 35	4.8	1.2

^{*} All values expressed as torr

Vessel	Mean	Range	S.D.	S.E.M.
Femoral Artery	94	91-97	1.4	0.4
Pulmonary Artery	51	37-65	7.9	2.0
Ant. Vena Cava	58	46-67	8.1	2.1
Post. Vena Cava	49	3 3- 63	9.3	2.4
Int Jugular Vein	52	34-72	16.0	4.1
Femoral Vein	46	32-62	14.4	3.7
Coronary Sinus	42	30-61	10.2	2.6

^{*} All values expressed as percent oxyhemoglobin

TABLE 4 $\label{eq:arterial} \mbox{Arterial and Venous O$_2$ Content Values* for Young Swine }$

	Vessel	Mean	Range	S.D.	S.E.M.	
•	Femoral Artery	15.4	14.0-16.6	0.90	0.23	
	Pulmonary Artery	8.5	5.7-11.2	1.46	0.39	
	Ant. Vena Cava	9.5	7.1-11.4	1.60	0.41	
	Post. Vena Cava	8.1	6.2-10.7	1.63	0.42	
	Internal Jugular	9.3	5.6-12.1	1.69	0.44	
	Femoral Vein	8.1	5.2-10.6	1.60	0.41	
	Coronary Sinus	6.9	4.4- 9.0	1.51	0.39	

^{*} All values expressed as m1 $O_2/100$ m1 blood

TABLE 5

Arterial and Venous pH Values* for Young Swine

Vessel	Mean	Range	s.D.	S.E.M.
Femoral Artery	7.399	7.373-7.415	0.0117	0.0030
Pulmonary Artery	7.335	7.306-7.408	0.0259	0.0067
Ant. Vena Cava	7.323	7.237-7.359	0.0303	0.0078
Post. Vena Cava	7.341	7.296~7.413	0.0331	0.0085
Int. Jugular Vein	7.328	7.280-7.371	0.0265	0.0069
Femoral Vein	7.302	7.232-7.343	0.0311	0.0080
Coronary Sinus	7.341	7.306-7.381	0.0243	0.0063

^{*} All values expressed in pH units

TABLE 6

Arterial and Venous P CO₂ Values* of Young Swine

Vesse1	Mean	Range	S.D.	S.E.M.	
Femoral Artery	47	42-54	3.5	0.9	
Pulmonary Artery	57	47-66	5.8	1.5	
Ant. Vena Cava	58	49-65	5.2	1.3	
Post. Vena Cava	57	46-67	5.9	1.5	
Int. Jugular Vein	58	48-68	6.3	1.6	
Femoral Vein	62	50-76	7.0	1.8	
Coronary Sinus	57	48-67	5.3	1.4	

^{*}All values expressed at torr

TABLE 7

Arterial and Venous Bicarbonate Values* for Young Swine

Vesse1	Mean	Range	S.D.	S.E.M
Femoral Artery	27.6	26.0-30.0	1.87	0.48
Pulmonary Artery	29.6	26.6-33.6	2.31	0.60
Ant. Vena Cava	29.5	26.6-34.0	2.17	0.56
Post. Vena Cava	29.8	27.1-34.8	2.17	0.56
Int. Jugular Vein	29.6	26.6-33.9	2.31	0.60
Femoral Vein	29.4	26.2-34.0	2.01	0.52
Coronary Sinus	29.8	27.3-34.8	2.32	0.60

^{*}All values expressed as mEq/liter

TABLE 8

Arterial and Venous Base Excess Values* for Young Swine

Vessel	Mean	Range	s.D.	S.E.M.
Femoral Artery	+2.8	+0.6 - +6.6	1.80	0.47
Pulmonary Artery	+3.9	+1.0 - +7.4	2.14	0.55
Ant. Vena Cava	+3.6	+1.0 - +7.8	2.01	0.52
Post Vena Cava	+4.3	+1.5 - +9.0	1.90	0.49
Int. Jugular Vein	+3.7	+1.4 - +7.6	2.02	0.52
Femoral Vein	+3.7	+1.1 - +7.5	2.08	0.54
Coronary Sinus	+4.7	+2.0 ~ +8.9	2.02	0.52

^{*} All values expressed as mEq/liter

TABLE 9a

Analyses of Variance: Evaluation of Differences in Blood Gas and Acid-Base Characteristics of Venous Blood Taken from Various Vascular Sites of Young Pigs

ANOVA

Variable	Mean Square	F-Ratio
PO ₂	176.0	15.18*
Oxygen Saturation	457. 6	13.33*
Oxygen Content	13.05	13.30*
pH	3.13×10^{-3}	10.69*
r co,	52.95	9.61*
Bicarbonate Conc	0.415	0.70
Base Excess Conc	2.400	4.76*

^{*}Significant difference at P \leq 0.05: $F_{5,70}$ =2.37

Newman-Keuls Evaluation of Differences in Blood
Gas and Acid-Base Characteristics of Venous
Blood Taken from Various Vascular Sites of Young
Pigs

Variable	PA	AVC	PVC	IJ	FV	CS
PO ₂	a	b	a	b	a	С
s o ₂	С	a	b	a	b	c
c 0 ₂	a	b	a	b	a	c
pН	ab	b	a	b	С	ab
P CO ₂	a	a	a	a	С	С
[HCO ₃]	a	a	a	a	a	a
[BE]	a	ab	С	ab	b	C

Abbreviations: PA= pulmonary artery, AVC= anterior vena cava, PVC= posterior vena cava, IJ= internal jugular, FV= femoral vein, CS= coronary sinus

Like letter in a given line indicates no significant ($P \le 0.05$) difference between vascular sites.

TABLE 10

Correlation Matrix for Arterial and Mixed Venous Blood

Gas and Acid-Base Variables of Young Swine

	Нф	P 0 ₂	P CO ₂	[HCO_]
		Femoral Art	ery	
P 02	-0.226			
P CO ₂	-0.474*	0.522*		
[HCO ³]	-0.126	0.517*	0.871*	
[BE]	-0.002	0.582*	0.858*	0.957*
		Pulmonary A	rtery	
PO2	0.287			
P CO ₂	-0.633*	-0.086		
[HCO ₃]	-0.134	0.088	0.842*	
[BE]	0.081	0.248	0.701*	0.972*

^{*}Significant interrelationship at P \le 0.05: df=13, r=0.441

TABLE 11

Correlation of Femoral Artery Blood Gas and Acid-Base

Variables to Ventilatory Variables of Young Swine

	F	F _I O ₂	v ' _T	v' _I
Нq	-0.265	-0.418	0.213	-0.036
P CO ₂	0.457*	0.890*	-0.425	-0.078
[HCO3]	0.405	0.465*	0.333	-0.049
PO2	0.224	0.927*	0.015	0.207

[†] Ventilatory variables: f= respiratory frequency in breaths/min; F₁O₂ = percentage O₂ inspired;

 $V_T' = tidal \ volume \ in \ ml/kg; \ and \ V_I' = inspiratory \ minute volume \ in \ ml/kg/min.$

^{*}Significant interrelationships at P \leq 0.05: df = 13, r = 0.441

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